measured resting MBF with positron emmission tomography using either O-15 water or N-13 ammonia and the myocardial response to Dob (2.5 to 40 μg/kg/min) with transesophageal echocardiography in 15 patients (14 men; age 61 ± 9 years) with resting ejection fraction < 45% (mean: 30 ± 10). Systolic wall thickening (SWT) and MBF were measured quantitatively in 8 anatomically matched myocardial segments in each patient. A total of 115 segments were available for analysis. SWT at rest ranged from −11% to 71% (normal > 35%) and MBF from 0.17 to 1.7 ml/g/min (normal ≥ 0.6). Segments with normal MBF had significantly greater contraction at rest compared to those with reduced MBF (SWT: 29.4 ± 19% vs. 14.8 ± 18%; $P < 0.0001$). Furthermore, the inotropic response to Dob was also significantly greater in regions with preserved MBF (increase in percent SWT: 28.6 ± 22% vs. 16.9 ± 16%; $P = 0.007$). A positive response to Dob (increase in percent SWT > 20%) was more likely in segments with normal MBF (51 of 72 [71%] vs. 14 of 43 [33%]; $P = 0.0001$). Thus, both resting myocardial contraction and the inotropic response to Dob are related to MBF. These findings suggest that reduced coronary blood flow is a determinant of myocardial contractile reserve in patients with CAD and LV systolic dysfunction.

907-55 Effect of Low-Density-Lipoprotein Apheresis on Coronary Anatomy and Regional Myocardial Blood Flow

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In patients with coronary artery disease (CAD) and hypercholesterolemia progression of the disease is common despite treatment with lipid-lowering medication. LDL-Apheresis over dextran sulphate columns is a very effective lipid-lowering therapy which might have an beneficial effect on coronary anatomy and physiology.

Methods: In a randomized study we compared the effect of biweekly LDL-apheresis and simvastatin versus simvastatin alone. Patients with total cholesterol ≥ 8 mmol/l and severe CAD were included in the study, 21 in the LDL-apheresis (L) group and 21 in the medication (M) group. Mean segment diameter (MSD) and minimal obstruction diameter (MOD) were assessed by QCA before and after 2 years of therapy. The regional myocardial blood flow was assessed by digital subtraction angiography after i.c. papaverin with video-densitometric calculation of the hyperemic mean transit time (HMTT).

Results: LDL-cholesterol decreased from 7.72 ± 1.96 mmol/l to a time averaged level of 2.95 ± 1.13 (−63%) in the L group and from 7.85 ± 2.34 mmol/l to 4.13 ± 1.58 mmol/l (−43%) in the M group. QCA revealed no differences in coronary anatomy either on a patient based or on a segment based comparison. Paired HMTT measurements were assessed in 43 regions in the L group and 35 regions in the M group. Baseline values for M and L group were not significantly different. In the L group HMTT decreased over 2 years in all regions from 3.35 ± 1.18 to 2.87 ± 0.82 s. ($p < 0.01$) versus an increase in the M group from to 2.95 ± 1.06 to 2.96 ± 0.90 (NS). The HMTTs of the LAD, RCX and RCA region contributed to the same extent in the final result.

Conclusions: LDL-apheresis compared to simvastatin alone lowered LDL-cholesterol significantly more. Both groups showed no change in coronary anatomy. However, regional myocardial blood flow improved in the L group. This functional enhancement is in accordance with previous reported results of excercise tests and may be a marker of recovery of endothelial function.

907-56 Recovery of Function After Revascularization Is Dependent on Preservation of Myocardial Blood Flow

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The utility of myocardial blood flow (MBF) for the evaluation of recovery of function after revascularization (rev) has not been well studied. In this study, we determined whether MBF differentiates asynergic regions that improve post-rev (viable) from those that remain abnormal post-rev (nonviable). We studied 9 pts with chronic CAD who underwent pre-rev positron emission tomography (PET) at rest using N-13 ammonia and F-18 deoxyglucose (FDG), pre- and post-rev gated magnetic resonance imaging (MRI) and radionuclide angiography. Mean LVEF at rest increased from 31 ± 8% pre-rev to 38 ± 11% post-rev ($p = 0.01$). Absolute MBF was computed for a single large region in which ammonia uptake was uniform and all other regions were then scaled by this MBF value. 2–4 MRI and PET transaxial slices were matched and analyzed per patient. Pre- and post-rev systolic wall thickening (SWT) was assessed visually by MRI and end-diastolic (ED) and end-systolic (ES) wall thickness was measured quantitatively in 5 regions per slice. From 145 regions studied, pre-rev SWT was normal in 89 (61%) and abnormal in 56 (39%) regions. In normal regions, mean MBF was 0.71 ± 0.17 and FDG uptake was 0.90 ± 0.23. Post-rev, SWT improved in 32 of 56 (57%) abnormal regions. PET and MRI data in post-rev improved and abnormal regions follow:

	PET		MRI			
	Blood Flow (ml/g/min)	FDG (% uptake)	ED (mm)		ES (mm)	
			pre	post	pre	post
Improved	0.73±0.2*	0.97±0.3*	7.1±2.6	8.2±2.6#	8.8±4.3	10.1±3.8#
Abnormal	0.57±0.2	0.77±0.2	4.7±1.5	5.1±1.8	6.1±2.0	6.1±1.9

*$p < 0.01$ (Improved vs Abnormal), #$p < 0.01$ (pre vs post-rev)

Regions with improved SWT post-rev had higher mean MBF and FDG values compared to abnormal regions. Although there was a linear relationship between MBF and FDG uptake in normal ($r = 0.6$, $p < 0.001$) and abnormal regions that did not improve post-rev ($r = 0.7$, $p < 0.001$), the relationship was nonlinear in regions that improved post-rev. The latter may be explained by the preferentially increased FDG uptake in ischemic but viable regions. These data suggest that recovery of function after rev is closely related to preservation of MBF at a level high enough to maintain glucose utilization and myocyte viability.

907-57 Value of Thallium-201 Viability Imaging for Evaluating Prognosis in Patients With Ischemic Left Ventricular Dysfunction

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Aim of this study was to determine the long term prognostic significance of maintained Thallium-201 uptake in patients with ischemic left ventricular dysfunction. We retrospectively analized 161 patients with angiographically documented coronary artery disease and reduced ejection fraction (0.35 ± 0.9) studied by Thallium-201 viability imaging (reinjection, late redistribution or rest studies). Thallium-201 uptake was quantified in 13 segments using normalized angular profiles and a viability cut-off of 54% of the peak; regional wall motion was assessed in the same segments using echocardiography. Average follow-up was 65 ± 50 months. Fifty-five/85 patients with dysfunctioning, mostly viable myocardium (> 50% of dysfunctioning segments) who did not undergo revascularization were more likely to experience hard events such as reinfarction (3 patients) or death (4 patients) when compared to the 30 successfully revascularized patients (1 death, no infarction). No significative difference was found between patients with dominance of cell death treated medically (76 patients) or revascularized (17 patients). These results suggest that Thallium-201 viability imaging appears to identify patients at increased risk of having reinfarction or death that have the most benefit from a revascularization procedure. Beyond short-term results, tissue viability as detected by Thallium-201 may provide important prognostic information guiding the management of patients with ischemically compromised myocardium.

907-58 Coronary Anatomy, Stress Myocardial Perfusion Defects, and Ambulatory Electrocardiographic Ischemia. What Is the Relationship? An ACIP Ancillary Study

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Stress myocardial perfusion imaging (SPECT), ambulatory electrocardiographic monitoring (AECG) and coronary arteriography (COR) were analyzed in 106 pts (77% men, age 61 ± 8 yrs (mean ± SD)) with > 50% coronary artery stenosis who had SPECT within 6 months of a screening AECG for enrollment in the asymptomatic cardiac ischemia pilot (ACIP) study. AECG, SPECT and COR were read independently in core labs. AECG ischemia was present in 65 pts (61%); 88 (83%) had SPECT perfusion defects ($p < 0.005$). Fifty pts were enrolled in ACIP; 56 were not. Most pts had perfusion defects by SPECT whether the AECG did (82%) or did not (85%) show ischemia. There were 2.7 ± 6.8 AECG ischemic episodes per 48 hours and SPECT defect size was 14 ± 14% of the left ventricle. By logistic regression methods, only ST segment depression on the exercise ECG during the SPECT study was associated with AECG ischemia ($p = 0.022$). No other relationship between SPECT and AECG results was found. COR explains these findings. SPECT defects were present with any ≥ 50% coronary stenosis, while AECG ischemia was related to proximal coronary artery lesions ($p = 0.04$). Neither SPECT defects nor AECG ischemia were related to plaque morphology or multivessel disease. Thus, SPECT and AECG are unrelated because SPECT defects are seen with any signficant coronary artery disease while AECG ischemia is related to proximal coronary artery lesions.